

KERATODERMIA BLENNORRHAGICA*

REPORT OF A CASE AND A SUGGESTION CONCERNING ITS NATURE

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The clinical condition known as Reiter's disease is of interest to venereologists, dermatologists, ophthalmologists, bacteriologists, and physicians. Some doubt exists regarding its cause though many theories have been put forward to explain it. Harkness (1950) gives a detailed account of the condition and reviews the literature thoroughly. He advocates separating the disease into two distinct types, firstly those of dysenteric origin (like Reiter's original case) to be called "dysenteric arthritis", and, secondly, those of venereal origin to be called "the non-gonococcal syndrome". He later admits, however, that both types are probably due to the same virus or pleuropneumonia-like organism, the portal of entry being the diseased mucous membrane of either the large intestine or the urethra. It should also be remembered that many workers consider that the disease bears no relation to sexual intercourse and is not connected with venereal disease.

The following case history, if the patient's story is believed, would support this view, but the main purpose of this report is to suggest a possible explanation (not new) for the skin lesions (keratoderma blennorrhagica) in Reiter's disease.

Case Report

Mr. N. J. S., aged 39, was first seen on September 13, 1950, when he attended a V.D. clinic. A tall, pale man, he limped into the consulting room complaining of a urethral discharge and pain in the ankles and feet.

History.—This was the fourth attack of this condition; the first had occurred in 1934 and had commenced with a urethral discharge, followed a week later by a rash confined to the soles and dorsum of the feet. Three weeks later swelling and pain developed in his knees, wrists, and elbows. He was admitted to hospital and recovery took place in 7 weeks. The second attack,

in 1938, was similar in all respects to the first but less severe and the patient recovered in 3 weeks. The third attack developed in 1942 whilst serving in the army. He was stationed in the U.K. and had never been abroad. The outbreak was similar in severity to the second and was identical in type to both previous attacks.

The present outbreak, his fourth, had commenced in August, 1950, with a urethral discharge, followed by the skin eruption and joint pains exactly as before, but the rash spread to involve his arms, trunk, and scalp. He described this attack as more severe than any of the previous ones. There was an additional symptom of a sensation of numbness in the legs and feet. Close questioning elicited a further symptom, which he stated had been present for 2 or 3 weeks before each attack and which disappeared as soon as the signs appeared. This consisted of an uncontrollable hunger. He stated that he awoke once or twice every night and was obliged to "raid the larder" to satisfy his ravenous appetite. Combined with the hunger was a sense of well-being and physical fitness which was unusual in him as, although rarely confined to bed or off work, he was by nature quiet and indolent. He denied any previous illnesses except colds. There was no family history of skin disease and between attacks he had no skin lesions of any kind. He had never suffered from gonorrhoea and had never complained of eye symptoms.

He was married in 1931, but separated from his wife in 1933, and then had a regular consort until he was re-united with his wife in 1936, since when he had lived a normal married life. He denied extra-marital risk except for the period 1933-36.

Examination

Genito-Urinary Tract.—A profuse, greyish-white, mucopurulent urethral discharge. The glans penis was congested and bright red in colour. There was no purulent balanitis (Fig. 1, overleaf).

Joints.—Visible swelling of the metacarpo-phalangeal joints of the index and middle fingers of the right hand, right knee joint, and both ankle joints. The circumference of the right knee was $14\frac{1}{2}$ in. compared with the left knee which was 13 in. The affected joints and feet were painful when moved and during weight bearing.

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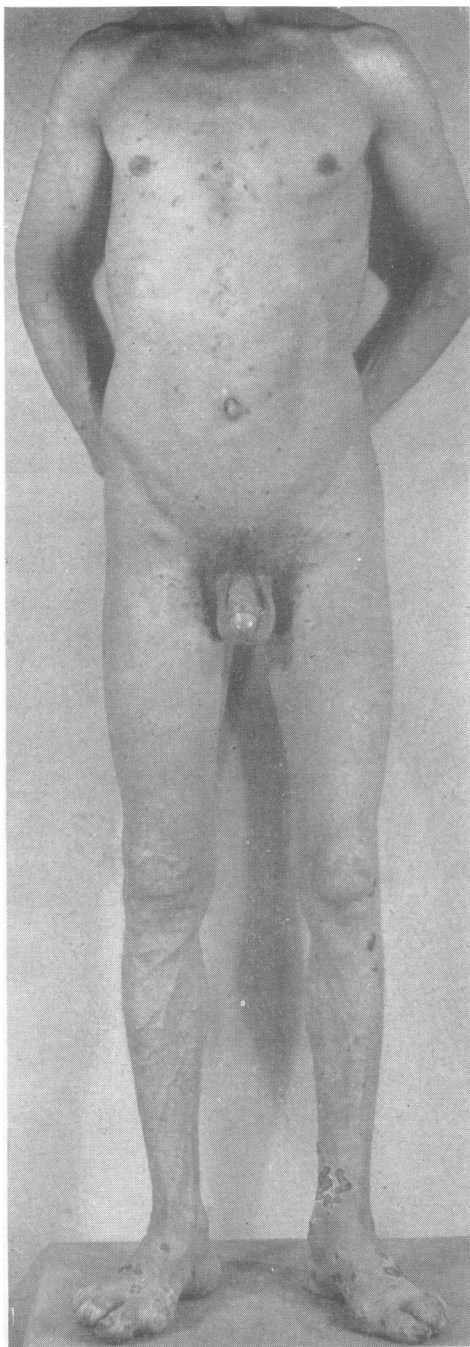


FIG. 1.—Scattered guttate lesions on trunk, groins, and left leg. Non-suppurative balanitis is also shown.

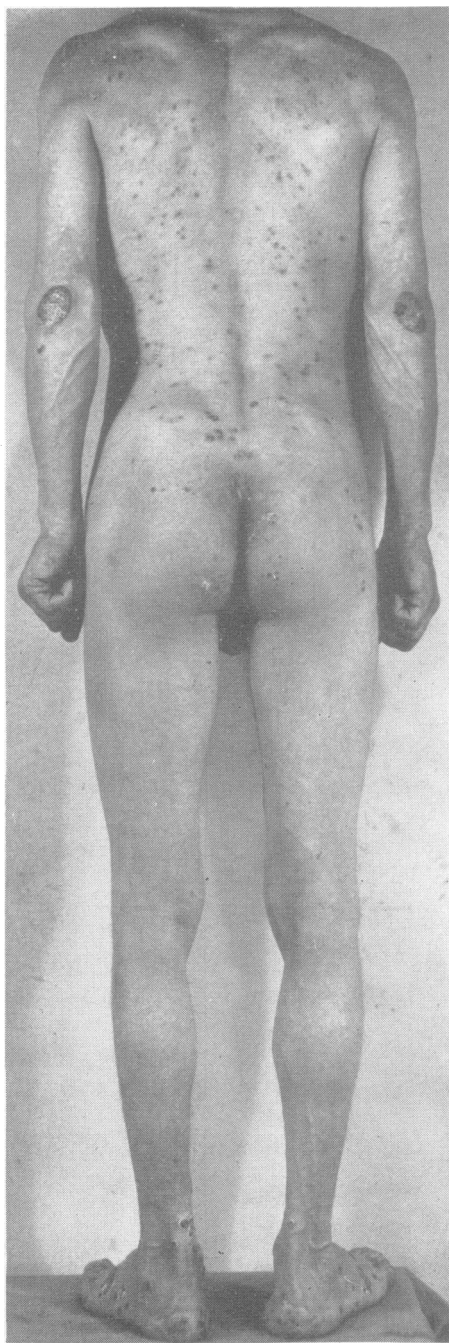


FIG. 2.—Many guttate lesions on buttocks and back, especially in lumbar region. Scaly plaques of psoriasis appear on the extensor surfaces of the elbows.



FIG. 3.—Palmar lesions showing keratotic papules. Note also scaly, heaped-up condition of peri-ungual and sub-ungual areas (thumbs).

the thenar eminences, were several red papules and nodules, many of which were "capped" with a white crust (Fig. 3). Three or four similar lesions were present on the backs of the hands. The finger nails were thickened at their free edge and for some distance proximally were raised from the nail bed and pitted. Periungually and subungually the skin was red and scaly with some desquamation. The appearance was that of "pustular" psoriasis (Fig. 4).

Groins.—Many closely set, scaly, guttate lesions were present in the groins and pubic area, while on the perineum and scrotum the lesions had become confluent and moist.

Skin

Scalp.—Scattered throughout the scalp were several discrete, red, scaly plaques indistinguishable from psoriasis capitis.

Trunk.—On the chest wall, abdomen, and back were many small red spots, some macular, some papular, and some covered with silvery scales resembling guttate psoriasis.

Arms.—On the extensor surface of both elbows were plaques identical with psoriasis vulgaris (Fig. 2). A few guttate lesions were present on the wrists.

Hands and Fingers.—On the palms, flexor surfaces of the fingers, and especially on

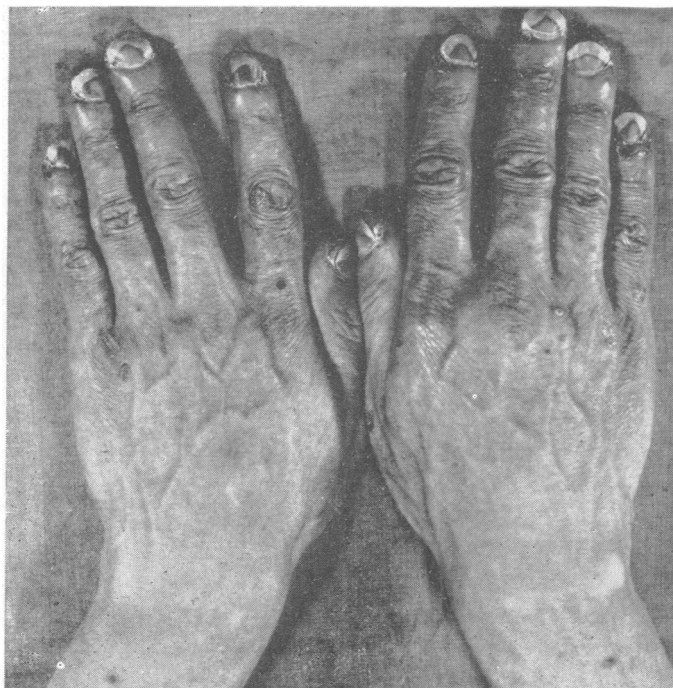


FIG. 4.—Swelling of metacarpo-phalangeal joints of index and middle fingers of right hand. Nails are separated and lifted from nail bed at distal portion of nail plate.

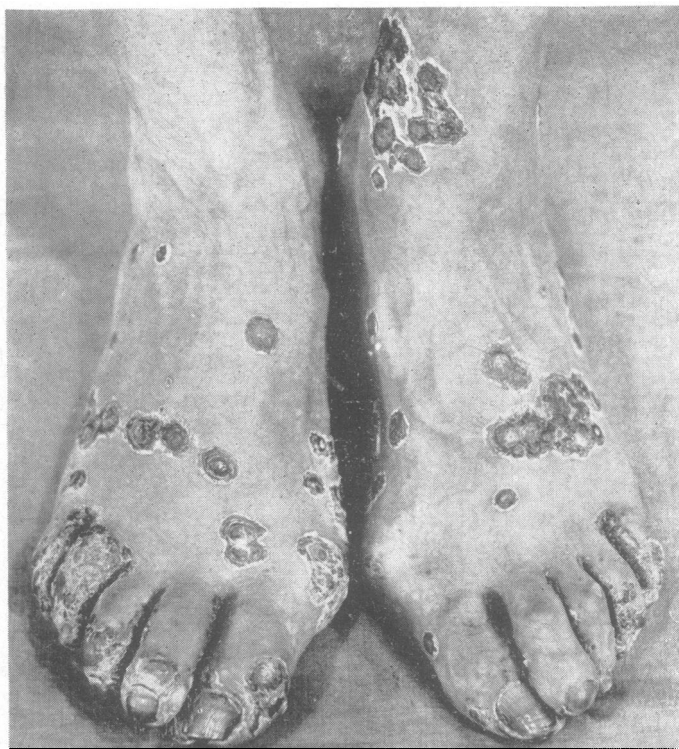


FIG. 5.—Moist patches on dorsum of feet with marked digital, inter-digital, and peri-ungual scaliness.

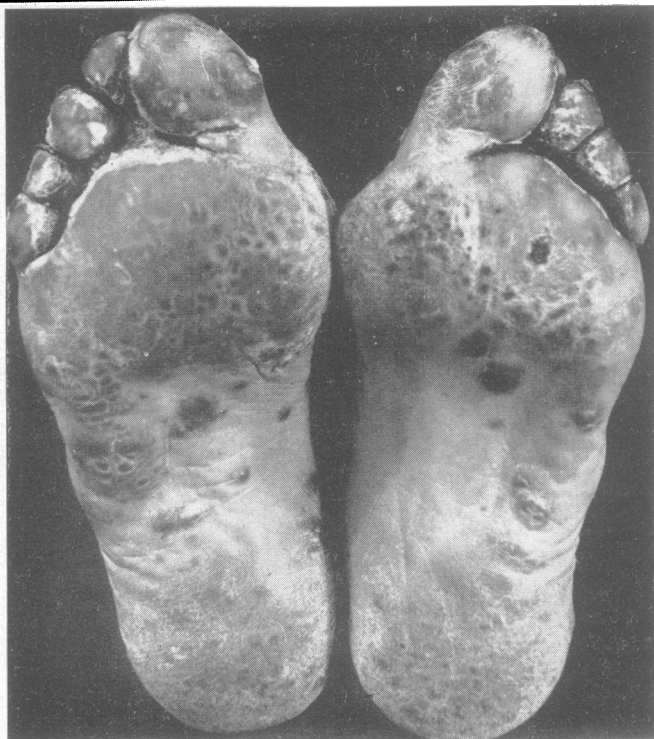
Legs, Feet, and Ankles.—On the outer side of the left leg and knee were several psoriasis-like lesions. On the inner side of the left ankle, the dorsum of both feet, and several toes were clearly defined discoid patches. Some were isolated, others so closely set that they merged, forming a serpiginous outline. They were red in colour and surrounded by a lip of undermined thickened skin similar to a ruptured bulla. The surface was dry with an attempt at scale formation, except on one or two of the smallest (and youngest) lesions where the surface was still moist. The nails, subungual, and periungual areas showed changes similar to those on the fingers (Fig. 5).

On the plantar surface of the feet, heels, and metatarsal heads there was marked hyperkeratosis through which could be seen many dark red papules surrounded by a white halo. Localized areas of hyperkeratosis of a nodular variety were present on the insteps (Fig. 6). The resemblance to "pustular" and "ruroidal" psoriasis was striking.

Eyes.—No abnormality was found.

Treatment.—He was admitted to hospital the next day and treated by rest in bed. No active therapy was given, except for a mild salicylic acid ointment for the more resistant psoriasiform skin lesions.

FIG. 6.—Classical keratoderma blennorrhagica with peeling and exfoliation commencing at base of toes on right foot.



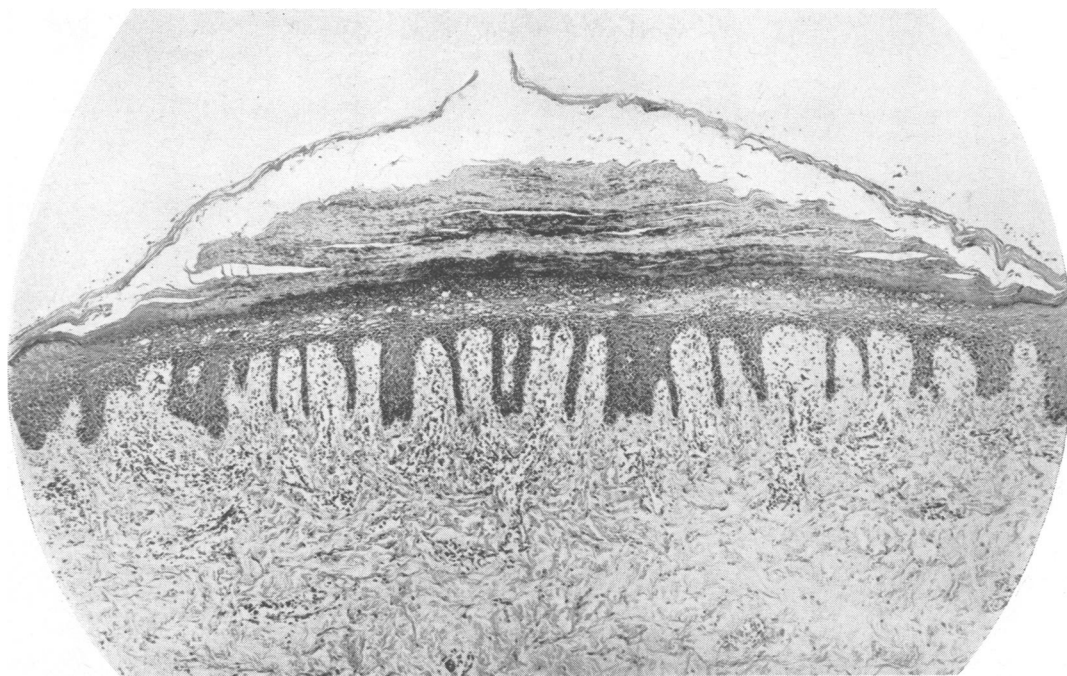


FIG. 7.—“Vesicular” stage showing intact stratum corneum and parakeratosis. Acanthosis is present with migration of leucocytes into the epidermis. A leucocytic infiltration is also present in the upper part of the corium.

Progress.—At first he was seen daily and rapid improvement was noticed at each visit. The urethral discharge disappeared first, then the joint swellings, and finally the skin lesions. The guttate-type spots faded first and the psoriasis vulgaris lesions (elbows) last. All signs and symptoms had gone by October 19, 1950, when the illness had lasted for 8 weeks. The skin lesions formed the most striking part of the syndrome, the youngest lesion was always a vesicle but differed from an ordinary vesicle by the presence of a red papule beneath. The vesicles soon ruptured leaving the red papule with a moist surface; those on the trunk then became dry and slowly faded; those on the arms, legs, and scalp became flat and covered with white scales identical with psoriasis; those on the palms and soles became markedly hyperkeratotic.

Investigations

Biopsy specimens were taken of three isolated skin lesions from the dorsum of the left foot and both wrists. All specimens were of approximately the same size and were removed whole. It was hoped to show the three distinct stages of the evolution of the cutaneous manifestations; vesicular, papular, and psoriasiform. Histological sections showed quite clearly that the three stages did, in fact, exist.

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(1) *Vesicular Stage.*—The stratum corneum is intact and forms a covering to a mass consisting of layers of parakeratotic horn cells, coagulated exudate, and enmeshed leucocytes. The rete cells are oedematous, and leucocytes are present in the inter-cellular spaces. There is acanthosis. A leucocytic infiltration is present in the papillary layer of the dermis. The blood vessels are dilated (Fig. 7).

(2) *Papular Stage.*—The stratum corneum has disappeared. Layers of parakeratotic horn cells rest on the rete from which the stratum granulosum is absent. There is a marked degree of acanthosis and papillary oedema. The degree of leucocytic infiltration is less (Fig. 8, overleaf).

(3) *Psoriasiform Stage.*—The mass of parakeratotic horn cells appears to have pressed the rete into the corium, forming a flat, plaque-like lesion instead of a raised papule. Acanthosis is still present (Fig. 9, overleaf).

Urethral smears taken on several occasions failed to reveal the gonococcus. Specimens of the urethral discharge and scrapings from the moist skin lesions were searched for *L. organisms* without success.

The patient's temperature varied between 98.2° and 100.4°F.

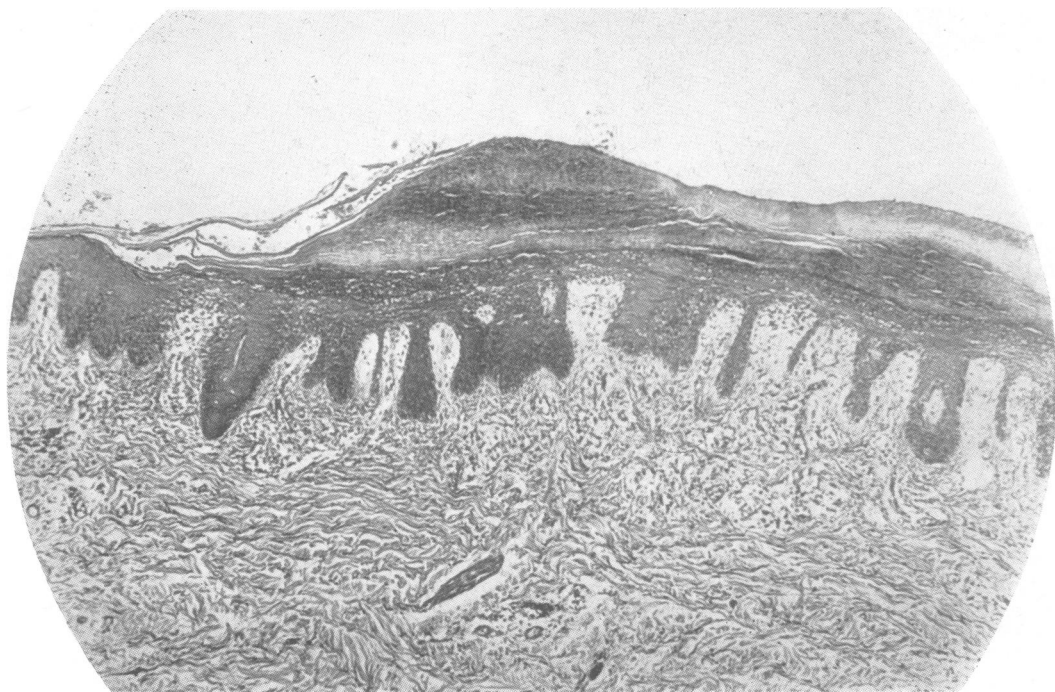


FIG. 8.—“Papular” stage showing absence of stratum corneum. The squama crust rests on acanthotic interpapillary processes.

A full blood count taken on September 15, 1950, showed :—

Haemoglobin	102 per cent. (Haldane)
Red blood cells	4,700,000 per c.mm.
Colour index	1.08
Total white cells	7,800 per c.mm.
Polymorphs	74 per cent.
Lymphocytes	19 per cent.
Monocytes	3 per cent.
Eosinophiles	4 per cent.

The Wassermann reaction and gonococcal complement-fixation test were negative.

The erythrocyte sedimentation rate was 78 mm. in the first hour and 94 mm. in the second hour.

A skiagram of the left hand on September 20, 1950, showed no abnormality.

Discussion

There can be little doubt that the diagnosis of Reiter's disease with keratoderma blennorrhagica is the correct one despite the absence of conjunctivitis. The abacterial urethritis, polyarthritis, and balanitis are sufficient evidence alone, and the skin eruption confirmed the opinion. Adamson (1920) suggested that keratoderma blennorrhagica was a form of psoriasis, and summarized his conclusions as follows :

(1) there are cases of arthropathic psoriasis in which the lesions on the palms and soles strikingly recall those of keratoderma blennorrhagica ;

(2) in many cases of keratoderma blennorrhagica there are eruptions on the trunk and limbs indistinguishable from psoriasis ;

(3) there are cases in which it is difficult to make a definite diagnosis between arthropathic psoriasis and keratoderma blennorrhagica ;

(4) there is a close similarity between the histopathology of the two diseases.

He then suggested that if the two conditions are identical then psoriasis may be caused by a micro-organism more or less closely related to the gonococcus. This view was put forward at a time when most workers considered that the gonococcus was usually responsible for keratoderma blennorrhagica. For instance, Lees and Percival (1931) state that “clinical evidence points definitely to the cutaneous eruption being a toxic manifestation due to a gonococcal infection . . .”. The majority of workers now would probably agree with Harkness (1950) that a virus is the more likely infective agent though there is often a concomitant gonococcal infection as well. Dermatologists are still unable to agree on the cause of psoriasis, but they often

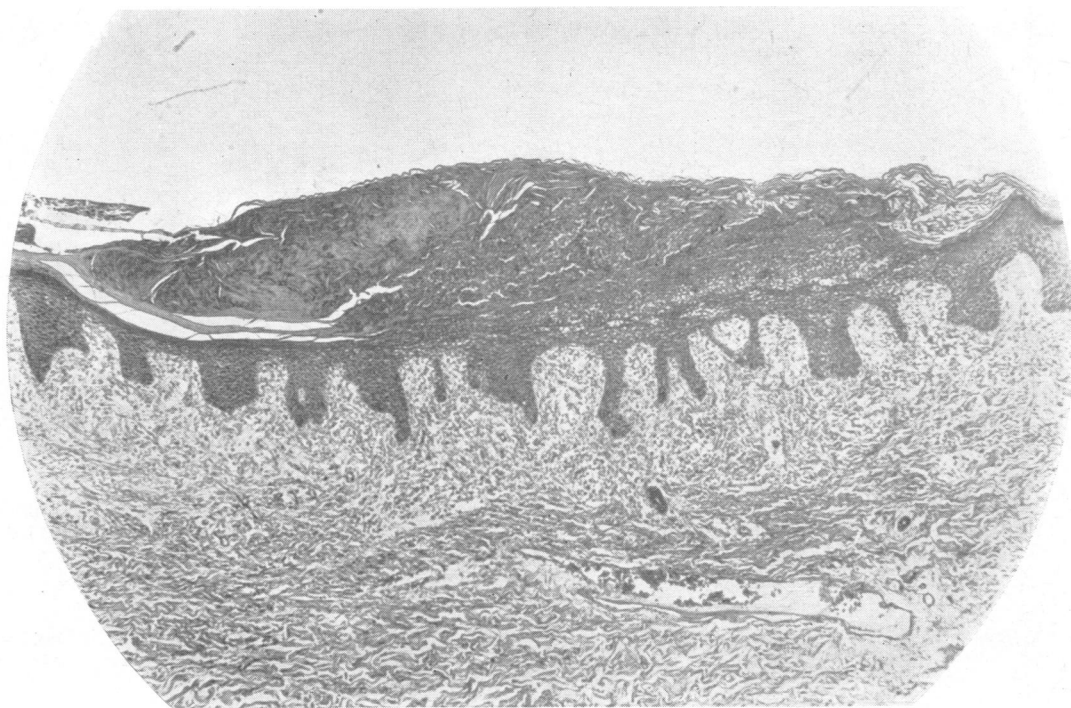


FIG. 9.—“Psoriasiform” stage. The squama crust, consisting of coagulated exudate, collections of degenerated leucocytes, and parakeratotic cells, appears to have pressed the interpapillary processes (rete pegs) into the corium.

observe the sudden and widespread exacerbations of the condition which occurs when the sufferer develops a bacterial infection of the throat, sinuses, lungs, or skin. It is not unusual to find that a patient's first attack of psoriasis occurs during or shortly after one of the infectious exanthemata, a severe boil, an attack of tonsillitis or sinusitis, influenza, bronchitis or impetigo. Subsequent outbreaks of psoriasis may then occur only when the patient again succumbs to a re-infection, or he may be left with a persistent chronic psoriasis which only spreads to distant parts of the body, like palms and soles, when a re-infection supervenes. It must not be thought that psoriasis is entirely due to infection; it is more probable that some constitutional or unknown factor is responsible for a person being a potential psoriatic, but an exciting super-added factor is required, acting like a trigger mechanism, before clinical manifestations appear. This provoking factor may be mental or physical shock, worry, trauma, or infection.

The histopathological differences in the two diseases are so slight that they could be accounted for by differences in the stage of development of the lesions examined, not by any fundamental

diagnostic detail. Personally I do not believe that any differences exist. May it not be then, that keratoderma blennorrhagica is psoriasis, provoked by the stimulus of a virus infection; the infection being at the same time responsible for the urethritis, arthritis, and conjunctivitis of Reiter's disease?

Conclusion

It would seem desirable to substitute the diagnosis of “Reiter's disease with psoriasis” for the term “keratoderma blennorrhagica”.

Summary

A case of Reiter's disease is reported. A suggestion is made that the cutaneous manifestations in such cases are in fact psoriasis.

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